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Air pollution in perspective: Health risks of air pollution expressed in equivalent numbers of passively smoked cigarettes

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ABSTRACT

Background: Although the health effects of long term exposure to air pollution are well established, it is difficult to effectively communicate the health risks of this (largely invisible) risk factor to the public and policy makers. The purpose of this study is to develop a method that expresses the health effects of air pollution in an equivalent number of daily passively smoked cigarettes.
Methods: Defined changes in PM2.5, nitrogen dioxide (NO_2) and Black Carbon (BC) concentration were expressed into number of passively smoked cigarettes, based on equivalent health risks for four outcome measures: Low Birth Weight (< 2500 g at term), decreased lung function (FEV_1), cardiovascular mortality and lung cancer. To describe the strength of the relationship with ETS and air pollutants, we summarized the epidemiological literature using published or new meta-analyses.
Results: Realistic increments of 10 μg/m^3 in PM2.5 and NO_2 concentration and a 1 μg/m^3 increment in BC concentration correspond to on average (standard error in parentheses) 5.5 (1.6), 2.5 (0.6) and 4.0 (1.2) passively smoked cigarettes per day across the four health endpoints, respectively. The uncertainty reflects differences in equivalence between the health endpoints and uncertainty in the concentration response functions. The health risk of living along a major freeway in Amsterdam is, compared to a counterfactual situation with 'clean' air, equivalent to 10 daily passively smoked cigarettes..
Conclusions: We developed a method that expresses the health risks of air pollution and the health benefits of better air quality in a simple, appealing manner. The method can be used both at the national/regional and the local level. Evaluation of the usefulness of the method as a communication tool is needed.

1. Introduction

Numerous studies have documented the adverse health effects of air pollution, even at levels well below the EU limit values (HEI, 2010; WHO, 2013; Beelen et al., 2015). In order to meet the European Union limit values, national and local authorities have to take action and attempt to reduce the emissions from mobile, domestic, agricultural and industrial sources. Because health effects of air pollution also occur below air quality limits, improvement of population health is an argument for further air pollution reduction policies. At the local level, policy measures are being implemented like congestion charges, low emission zones, increased parking rates. Policy makers need to "sell" the (often) expensive and restrictive measures to the public and create enough political support to implement them. Insight in the health impact of local air quality enhances acceptance and adoption of preventive measures (Briggs and Stern, 2007). This requires effective communication with the public about the health risks of local air pollution and the health benefits of improved air quality. However, it is difficult for local policy makers and health professionals to effectively communicate about the health risk of air pollution (Slovic, 1999; Weber, 2006; Bickerstaff and Walker, 2001).

One way to express health risks of local sources is by means of a risk quotient (relative risk or odds ratio) but this does not necessarily reflect perception of risks in a population, since perception is only partly based on scientific information (Slovic, 1999; Weber, 2006, Stewart et al., 2010). Alternatively, excess mortality

Abbreviations: BC, black carbon; CRF, concentration response function; ETS, environmental tobacco smoke; FEV1, Forced Expiratory Volume in 1 s; LBW, Low Birth Weight (Birthweight ≪ 2500 g after 37 weeks of gestation); IHD, ischaemic heart disease; NO_2, nitrogen dioxide; OR, odds ratio; PM2.5, particles smaller than 2.5 μm; PM10, particles smaller than 10 μm; RAP, risk advancement period; RR, relative risk; YLL, years of life lost

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risks resulting from exposure to local air pollution can be translated into years of life lost (Brunekreef et al., 2007) or risk-advancement period (Brenner et al., 1993), where years of life lost (YLL) are extrapolated into 'real age'. In a recent study, Geelen et al. (2013) reported that 'real age' of an individual increased with up to 36 days near the highway in the Moerdijk area in the Netherlands, compared to the background concentration. The impact of the implementation of a low emission zone in Rome was expressed as the gain in life expectancy: 921 years per 100.000 inhabitants, on average 3.4 days per person (Cesaroni et al., 2012). Modeled benefits of the London congestion charging zone was 183 years of life per 100.000 (0.7 days per person) inhabitants in the charging zone wards (Tonne et al., 2008). The effectiveness of communicating risk advancement periods, or years of life lost or gained due to air pollution to the public and policy is generally not evaluated. Yet, effective communication is important for the above mentioned reasons.

In a previous Dutch study, the impact of local traffic on Black Carbon (BC) concentration was translated into YLL based on the relation between BC exposure and life expectancy described by Janssen et al. (2011) and presented graphically at building-level for all major roads in the densely populated provinces North- and South-Holland (van der Sluis et al., 2012). The usefulness of this information for local policy makers and governors was evaluated in interviews. They were insufficiently able to interpret the effect on YLL and were unanimous in their wish for a simple, appealing comparison of the health risks of local air pollution with other, well-known risk factors. Risk factors that were mentioned were: passive or active smoking; obesity; unhealthy diet; traffic accidents (van der Sluis et al., 2012). Recently, Kelly and Fussell (2015) stressed that in order to increase public awareness, communication about the health risks of air pollution should be blatant and put in the context of other public health risks such as passive smoking. The principle of risk comparison for better communication of risk to the public was used earlier, for example in the Global Burden of Disease project, where the risks of a large number of risk factors including outdoor air pollution and passive smoking have been compared globally and for various regions of the world (Lim et al., 2012; Forouzanfar et al., 2015).

Pope et al. (2009) and Smith and Peel (2010) used the inhaled dose of PM2.5 from active smoking, household air pollution (indoor biomass and coal burning), passive smoking and outdoor air pollution to compare deaths due to cardiovascular disease. Due to the non-linear shape of the exposure-response relationship, much larger health benefits may occur at the lower end of the dose spectrum (Smith and Peel, 2010). The inhaled dose of PM2.5 is more than 300 fold higher for the average smoker than for the average passive smoker (Pope et al., 2009; Öberg et al., 2010). Inhaled PM2.5 doses for outdoor air pollution and passive smoking are comparable. As a consequence, the health effects of outdoor air pollution can more meaningfully be compared with those of passive smoking than of active smoking.

The aim of this study is to express the health effects of air pollution in equivalent amounts of passive smoking. We compared health risks of air pollution with passive smoking because both risk factors are comparable with respect to the exposure route (inhalation); have similar health effects both resulting from a complex mixture of particles and gases; and exposure to both air pollution and environmental tobacco smoke (ETS) is largely involuntary.

A simple tool is developed based on the relative risks describing the association between exposure to ETS and three key air pollutants: particulate matter with aerodynamic diameters < 2.5 μm (PM2.5), nitrogen dioxide (NO_2) and Black Carbon (BC) and four health outcomes (Low Birth Weight, lung function decrements in children, cardiovascular mortality and lung cancer).

We illustrate the method by expressing the health effects of living near a freeway, the health gain of a local traffic measure and the health effects of the emissions of a steel factory into equivalent amounts of exposure to ETS.

2. Material and methods

2.1. Selection of health outcomes

First, we searched the air pollution epidemiology literature for health outcomes with the most secure evidence of an association with PM2.5, NO_2 and Black Carbon (BC). PM2.5 is the preferred air pollution indicator for health impact assessment at the national or regional level. NO_2 and BC are the preferred air pollution indicators for health impact assessment at the local level, in situations where traffic is the primary source of air pollution. We made use of published (systematic) reviews, supplemented with more recent key studies.

Second, we searched the passive smoking epidemiology literature to select health outcomes with the most secure evidence of an association with ETS exposure. We made use of WHO and Surgeon General Reports and published (systematic) reviews.

Next, we selected health outcomes with the most secure evidence of an association for both the relation with ETS exposure and air pollution. We further aimed at including health outcomes reflecting adult and childhood health responses to evaluate differences in the ratio of air pollution and ETS health effects. We finally included four health outcomes:

1. Low Birth Weight (LBW) defined as a birth weight less than 2500 g after 37 weeks of gestation.
2. Lung function (FEV1) in school aged children.
3. Lung cancer.
4. Cardiovascular mortality.

2.2. Exposure-response functions for the relation between ETS exposure and health outcomes

Continuous data on ETS exposure (number of cigarettes) is rarely available in epidemiological studies. Some epidemiological studies have a more quantified assessment of ETS exposure, often classified into "low or moderate ETS exposure" or "moderate to high exposure". However, the cut-off points for the different exposure categories differ between studies. Therefore, meta-analyses such as the WHO's Global Burden of disease related to ETS (Öberg et al., 2010) provide estimates based on dichotomous exposure classification (presence / absence of ETS in the home or at work). Table 1 provides an overview of the risk estimates for ETS exposure for the selected health outcomes.

2.3. Assessment of ETS exposure

The risk estimates in Table 1 are based on dichotomous exposure classification. However, an estimate of the average daily residential exposure to ETS is essential to express air pollution health effects in an equivalent amount of cigarettes smoked. Based on estimates from the WHO for smokers in the US and North-West Europe, we assume an average of 14 daily cigarettes (Öberg et al., 2010).

The average daily *residential* exposure was estimated following a (modified) approach by Nazaroff and Singer (2004). This is based on the assumption that the average smoker consumes half of their daily cigarettes indoors at home, which follows from an expectation that habitual smokers will consume cigarettes at a roughly uniform rate throughout the hours that they are awake. People in

developed countries spend approximately 65% of their time indoors at home (Leech et al., 2002; Brasche and Bischof, 2005) which, allowing for 8 h sleep per night, implies that about half of the daily awake hours are spent indoors at home.

Therefore, the daily average indoor cigarette exposure is estimated as:

$$N = 0.5 \times 14 = 7 \text{ passively smoked cigarettes/day}$$

This estimate is used to represent ETS exposure in relation to risk of cardiovascular mortality, lung cancer and Low Birth Weight for infants of non-smoking mothers.

Children who are exposed to parental smoking can have one or two smoking parents. Information about the number of smoking parents in the household is scarce. Based on a large national survey in England it was estimated that among parents who smoked in two parent households, 35% lived with an adult who also smoked (Belvin et al., 2015). In a smaller survey in the Netherlands 32% of children exposed to parental smoking reported that both parents smoke (Schuck et al., 2013). We assume that 32% of children, exposed to parental smoking have two smoking parents.

Consequently, the average indoor cigarette exposure for children exposed to parental ETS in relation to lung function decrements (FEV1) is estimated as:

$$N = 0.5 \times 14 \times 1.32 = 9 \text{ passively smoked cigarettes/day}$$

where 1.32 represents the average number of smoking parents for children exposed to parental smoking.

Furthermore, the assumption is that the relation between the number of passively smoked cigarettes smoked and health outcomes is linear.

2.4. Concentration response functions for the relation between air pollution and health outcomes

Concentration response functions (CRFs) describing the strength of the association between a defined change in air pollution concentration and health outcomes were derived from recent reviews. If recent key studies were published but meta-analyses were lacking, new meta-analyses were performed for the present study (described in the Supplemental material). Faustini et al. (2014) provides Relative Risks (RRs) for the association between NO_2 and PM2.5 and cardiovascular mortality. For BC the association with cardio-respiratory mortality is established in meta-analyses (Smith et al., 2009). Since cardiovascular deaths are the large majority of cardio-respiratory deaths, we used the Relative Risks reported for cardio-respiratory mortality for cardiovascular mortality.

Table 2 presents an overview of the risk estimates and the associated 95% confidence intervals for the four health outcomes.

2.5. Comparison of health risks

2.5.1. Comparison of risk estimates

The comparison is based upon the risk estimates reported in Tables 1 and 2. First, for each combination of component and health outcome the regression coefficient (β_{airpol}) per 1 μg/m^3 was calculated. For the dichotomous health outcomes, this was calculated from the RR and the corresponding change in concentrations:

$$\beta_{airpol} = \frac{\ln(RR)}{\Delta concentration} \quad (1)$$

with Δ concentration = 10 μg/m^3 for PM2.5 and NO_2 and 1 μg/m^3 for BC.

The regression coefficient per cigarette ($\beta_{cigarette}$) was calculated from the RR for ETS exposure and the assumed number of daily passively smoked cigarettes this exposure represents:

$$\beta_{cigarette} = \frac{\ln(RR)}{\text{assumed number of cigarettes}} \quad (2)$$

where the assumed number of passively smoked cigarettes is 9 for children (in relation to lung function decrements) and 7 for adults (in relation to LBW, cardiovascular mortality and lung cancer).

The ratio R of the resulting regression coefficients represents the number of passively smoked cigarettes that is equivalent to a 1 μg/m^3 increase in pollutant concentration for a specific health outcome:

$$R = \frac{\beta_{airpol}}{\beta_{cigarette}} \quad (3)$$

Table 1
Risk estimates for ETS exposure (compared to no ETS exposure) in relation to health outcomes.

Outcome	Description	Risk estimate[a] (95% CI)	Exposure	Reference
Low Birth Weight	Prevalence of LBW < 2500 g at term	1.38 (1.13–1.69)	Any exposure at work or at home; in non-smoking mothers	Windham et al., 1999
Reduced lung function in school aged children	Percentage reduction in Forced Expiratory Volume in 1 s (FEV1)	1.4% (1.0–1.9%)	Either parent	Cook et al., 1998
Ischaemic heart disease (IHD) mortality (> 15 yrs)	IHD mortality > 15 yrs	1.27 (1.19–1.36)	At home or at work; in non-smokers	Surgeon General Report, 2006; Öberg et al., 2010
Lung cancer (> 15 yrs)	Incidence	1.21 (1.13–1.30)	At home; in non-smokers	Surgeon General Report, 2006

[a] Relative risks for LBW, lung cancer, IHD mortality, and percentage decrement for lung function.

Table 2
Risk estimates (95% CI) for PM2.5, NO_2 and BC concentration and the established health outcomes.

	NO_2 (per 10 μg/m^3)	BC (per 1 μg/m^3)	PM2.5 (per 10 μg/m^3)
Low Birth Weight (< 2500 g)	1.06 (1.00–1.11)[a]	1.16 (0.93–1.44)[a]	1.19 (1.00–1.42)[a]
Percentage lung function decrement in school aged children (FEV1)	0.57% (0.0–1.14%)[a]	1.3% (0.3–2.2%)[a]	1.5% (−0.3–3.2%)[a]
Cardiovascular mortality	1.13 (1.09–1.18)[b]	1.11 (1.03–1.19)[c]	1.20 (1.09–1.31)[b]
Lung cancer	1.05 (1.02–1.08)[a]	1.04 (0.97–1.12)[a]	1.09 (1.04–1.14)[d]

[a] Based on new meta-analysis (described in Supplemental material).
[b] Faustini et al., 2014.
[c] Smith et al., 2009.
[d] Hamra et al., 2014.

Both the risk estimates for air pollution and ETS exposure are estimated with error. For each combination of pollutant and health outcome, the standard error of the ratio R taking both standard errors into account was calculated from the relation:

$$\left(\frac{SE_R}{R}\right)^2 = \left(\frac{SE_{airpol}}{\beta_{airpol}}\right)^2 + \left(\frac{SE_{cigarette}}{\beta_{cigarette}}\right)^2 \quad (4)$$

With

SE_R = standard error of the ratio R.

$SE_{cigarette}$ = standard error of the regression coefficient per passively smoked cigarette.

SE_{airpol} = standard error of the regression coefficient for a 1 μg/m^3 increase in concentration.

From the ratio R, the equivalent number of passively smoked cigarettes ("passive cigarette-equivalence") can be calculated for any change (delta) in pollutant concentration according to the formula:

$$\text{passive cigarette equivalence} = \Delta\text{concentration} \times R \quad (5)$$

With the associated standard error:

$$SE_{passive\ cigarette\ equivalence} = \Delta\text{concentration} \times SE_R \quad (6)$$

After inspecting differences in passive cigarette-equivalents for the four health outcomes per pollutant, arithmetic and weighted average passive cigarette-equivalents were calculated.

2.5.2. Arithmetic average and uncertainty

The pooled uncertainty in the estimation of the arithmetic average number of passively smoked cigarettes per pollutant was calculated according to formula (7):

$$(SE_{pooled\ per\ pollutant})^2 = \frac{1}{n^2}\ ((SE_{LBW})^2 + (SE_{LF})^2 + (SE_{CVD})^2 + (SE_{lung\ cancer})^2) \quad (7)$$

where n = the number of evaluated health effects per pollutant (n = 4).

If more than one pollutant is evaluated, the pooled standard error for all pollutants is calculated with the same formula:

$$(SE_{pooled})^2 = \frac{1}{n^2}((SE_{pooled\ for\ pollutant\ 1})^2 + .. + (SE_{pooled\ for\ pollutant\ n})^2)$$

where n = the number of evaluated pollutants (n = 2 or 3).

2.5.3. Weighted average and uncertainty

The weighted average passive cigarette-equivalence was calculated per pollutant using the inverse of the variance for each health outcome as weight:

$$\text{weight} = 1/(SE_{LBW})^2 \text{ for Low Birth Weight,}$$

$$\text{weight} = 1/(SE_{LF})^2 \text{ for reduced lung function etc.}$$

Endpoints with more precisely established health effects are weighed more. The uncertainty in the estimation of the weighted average passive cigarette-equivalence was calculated as the inverse of the square root of the sum of the weights (DerSimonian and Laird, 1986). If more than one pollutant is evaluated, the pooled uncertainty was calculated in the same way.

2.6. Relevant pollutant(s) to be evaluated?

Depending on the situation and the source of air pollution, PM2.5, BC and/or NO_2 may be the most relevant air pollutants to evaluate. For instance, a local traffic measure generally has a large impact on NO_2 and BC concentration but less impact on the PM2.5 concentration. In contrast, a steel factory affects local PM mass concentrations much more than NO_2 and/or Black Carbon concentrations. In each situation, the most affected pollutant(s) is the most relevant pollutant to evaluate.

2.7. Sensitivity analyses

The estimated passive cigarette-equivalency highly depends on the assumed number of daily passively smoked cigarettes indoors at home. To illustrate the impact of the assumed number of daily that a smokers smokes indoors at home on the passive cigarette-equivalency, we performed sensitivity analyses with three alternative scenarios:

1. Assuming that smokers do not consume half of their daily cigarette consumption indoors at home, as suggested by Nazaroff and Singer (2004) but only 25%.
2. Assuming a daily average of 18 smoked cigarettes, taking into account that people tend to underestimate their cigarette consumption in questionnaires, and based on tobacco sales rates in the Netherlands (Nazaroff and Singer, 2004; Matt et al., 2004; Forey et al., 2014).
3. Assuming that children are exposed to equal amounts of indoor smoked cigarettes as adults.

3. Results

Table 3 expresses the health risks of exposure to PM2.5, NO_2 and BC into equivalent numbers of passively smoked cigarettes, including its uncertainty. The estimates are presented for an increment of 10 μg/m^3 in NO_2 and PM2.5 concentration and 1 μg/m^3 in BC concentration. These increments are commonly used to express relative risks of these air pollutants. The meaning of these contrasts depends on the setting to which the method is applied. Realistic examples will be shown below.

Table 3 shows that the passive cigarette-equivalent estimates per pollutant for the 4 different health outcomes vary within a factor two to three for NO_2 and $PM_{2.5}$ and more for BC. Most estimates per pollutant agree reasonably when the uncertainty is taken into account. The comparison between the three pollutants depends on the selected increments and is more meaningfully looked at in the examples below. The variability in passive cigarette-equivalents is likely due to a combination of the wide range of studies providing relative risks for different pollutants and different health outcomes.

Exposure to air pollution is equivalent to a relative large number of passive cigarette-equivalents in relation to lung function decrements, and a relatively small number of passive cigarette-equivalents in relation to lung cancer. The uncertainty is reflecting the number of studies and consistency between studies,

Table 3
Health risks of NO_2, BC and PM2.5 expressed into equivalent numbers of passively smoked cigarettes.[a]

	NO_2	BC	PM2.5
Low Birth Weight	1.3 (0.7)	3.2 (2.7)	3.8 (2.3)
Percentage lung function decrement in school aged children (FEV1)	3.7 (2.0)	8.4 (3.3)	9.6 (5.8)
Cardiovascular mortality	3.6 (0.8)	3.1 (1.1)	5.3 (1.5)
Lung cancer	1.7 (0.6)	1.4 (1.4)	3.2 (1.0)
Arithmetic mean	2.5 (0.6)	4.0 (1.2)	5.5 (1.6)
Weighted mean[b]	2.0 (0.4)	2.8 (0.8)	4.0 (0.8)

[a] Standard error in parentheses.

[b] Weighted mean is calculated with the inverse of the variance as weight for the four effect estimates.

and is largest for lung function decrements. The weighted mean estimates are somewhat lower than the arithmetic mean estimates, since smaller estimates of passive cigarette-equivalents tend to have smaller standard errors and consequently, contribute more to the weight. The aim of this tool is to enable local policy makers and health professionals to effectively communicate about the health risk of air pollution. Therefore only arithmetic mean estimates are presented in the examples below, which are more easy-to-understand to the public than weighted mean estimates. This is supported by the idea that the full uncertainty is broader than the statistical uncertainty expressed in the standard error.

The Excel sheet used for the calculations is available in the online supplement. The results of the comparison will be illustrated with three examples.

Example 1. Living along the A10-ring freeway in Amsterdam.

One of the roadside stations of the Amsterdam Air Quality Monitoring Network is located directly along the A10-West freeway with a traffic intensity of 146,000 vehicles per day. Six story high apartment buildings are located just behind the measurement station and at the other side of the road, creating a situation resembling a street canyon.

We assumed that the exposure of the people living in the apartment buildings is equal to the annual mean concentrations measured, which is probably a slight overestimation. Since the freeway traffic has the largest impact on NO_2 and BC concentration, the health impact of those two pollutants are evaluated. In 2014, annual average NO_2 and BC concentrations of 51.5 $\mu g/m^3$ and 2.6 $\mu g/m^3$ were measured at this station, respectively. Those exposure levels are compared to a hypothetical counterfactual situation with annual mean NO_2 and BC of 10 $\mu g/m^3$ and 0.2 $\mu g/m^3$ respectively. Those levels are broadly representative of air quality in non-urban areas in Northern Scandinavia, the cleanest part of Europe. The difference in concentration is 41.5 and 2.4 $\mu g/m^3$ for NO_2 and BC, respectively (Table 4).

The arithmetic mean estimate of passive cigarette-equivalents per exposure metric are 10.6 (2.4) for NO_2 and 9.6 (2.8) for Black Carbon, with an overall-average of 10.1 (1.8) passive cigarette-equivalents.

Example 2. Evaluation of a local traffic measure in a busy street in The Hague.

Different routing of traffic in the Hague resulted in a 50% decrease in traffic intensity in a busy inner city road, which was 17,000 vehicles per day before the intervention (Boogaard et al., 2013). Air quality measurements, performed before (2008) and after (2010) implementation of the measure, revealed that this resulted in a decline of the traffic contribution of 1.4 $\mu g/m^3$ and 11.7 $\mu g/m^3$ for BC, and NO_2, respectively (Boogaard et al., 2013).The health benefits of those reductions in concentration, expressed in cigarette-equivalents, are presented in Table 5.

Table 4
Health risks of living along the Amsterdam freeway expressed in equivalent numbers of passively smoked cigarettes.[a]

	NO_2 $\Delta 41.5$ $\mu g/m^3$ [b]	BC $\Delta 2.4$ $\mu g/m^3$ [b]	Overall estimate
Low Birth Weight	5.3 (2.7)	7.6 (6.4)	
Percentage lung function decrement in school aged children (FEV1)	15.2 (8.2)	20.1 (8.0)	
Cardiovascular mortality	14.9 (3.5)	7.3 (2.7)	
Lung cancer	7.0 (2.5)	3.5 (3.3)	
Arithmetic mean	10.6 (2.4)	9.6 (2.8)	**10.1 (1.8)**

[a] Standard error in parentheses.
[b] Compared to a counterfactual 'clean' situation.

Table 5
Health benefits of 50% less traffic in a busy street expressed in equivalent numbers of passively smoked cigarettes.[a]

	NO_2 $\Delta 11.7$ $\mu g/m^3$	BC $\Delta 1.4$ $\mu g/m^3$	Overall estimate
Low Birth Weight	1.5 (0.8)	4.4 (3.7)	
Percentage lung function decrement in school aged children (FEV1)	4.3 (2.3)	11.7 (4.7)	
Cardiovascular mortality	4.2 (1.0)	4.3 (1.6)	
Lung cancer	2.0 (0.7)	2.0 (1.9)	
Arithmetic mean	3.0 (0.7)	5.6 (1.6)	**4.3 (0.9)**

[a] standard error in parentheses.

Table 6
Health risks of the emissions from a steel factory expressed in equivalent numbers of passively smoked cigarettes.[a]

	PM2.5 $\Delta 5.2$ $\mu g/m^3$
Low Birth Weight	2.0 (1.2)
Percentage lung function decrement in school aged children (FEV1)	5.0 (3.0)
Cardiovascular mortality	2.8 (0.8)
Lung cancer	1.6 (0.5)
Arithmetic mean	**2.9 (0.9)**

[a] Standard error in parentheses.

Table 7
Results of sensitivity analyses with different assumptions about the number of indoor smoked passive cigarettes equivalents.[a]

	Daily residential exposure of adults + unborn babies	Daily residential exposure of children	Overall estimate Example 1	Overall estimate Example 2	Overall estimate Example 3
Baseline	7	9	10.1 (1.8)	4.3 (0.9)	2.9 (0.9)
Scenario 1	3.5	4.5	5.0 (0.9)	2.1 (0.4)	1.4 (0.4)
Scenario 2	9	11	12.7 (2.3)	5.4 (1.1)	3.6 (1.1)
Scenario 3	7	7	9.1 (1.6)	3.8 (0.8)	2.6 (0.7)

Baseline: as presented in paper; scenario 1: assuming that 25% of the total number of cigarettes is smoked indoors at home instead of 50%; scenario 2; assuming a higher number of 18 daily smoked cigarettes (based on tobacco sales rates in the Netherlands); scenario 3: assuming that children and adults are equally exposed

[a] Standard error in parentheses.

The arithmetic mean estimate of passive cigarette-equivalents per exposure metric was 3.0 (0.7) for NO_2 and 5.6 (1.6) for BC. Overall, different routing of traffic and the resulting decline of 50% in traffic intensity resulted in improved air quality, comparable to avoiding residential exposure to ETS from 4.3 (0.9) cigarettes per day.

Example 3. Evaluation of the emissions from a steel factory.

In the Netherlands, a large steel factory contributes to particulate air pollution in the surrounding areas. Based on modeled emission of primary particles in the year 2000, it was estimated that the steel factory contributes 7.9 $\mu g/m^3$ to the annual mean PM10 concentration in the highest exposed communities (Ameling et al., 2014). Since PM mass concentrations are most affected by the steel factory's emissions, PM mass is the most relevant metric to evaluate in this example. Assuming a PM2.5/PM10 ratio of 0.66 (Cyrys et al., 2003), this corresponds to a PM2.5 concentration of 5.2 μg/m3. Table 6 presents the results.

Table 6 demonstrates that the health effects of the emissions from a steel factory are equivalent to residential exposure to ETS from 2.9 (0.9) cigarettes per day.

3.1. Sensitivity analyses

Table 7 presents the results of the sensitivity analyses presented in the method section.

Fewer passively smoked cigarettes in the home result in a higher regression coefficient per passively smoked cigarette and thus in a lower estimate of passive cigarette equivalents associated with a defined change in air pollution. The number of passively smoked cigarettes is linear related to the passive cigarette-equivalency, which is illustrated in Table 7. Assuming that 25% instead of 50% of the daily cigarette consumption is smoked indoors at home (scenario 1) reduces the passive cigarette-equivalency with a factor 2. Table 7 illustrates that in all scenarios, the health effect of 'living near the freeway' (Example 1) is slightly higher than the health risk of 'living with a smoker', regardless of the assumed number of indoor smoked cigarettes. The health benefits of 'a 50% decline in traffic intensity' (Example 2) are in all scenarios roughly equivalent to the health benefits of 'living with a smoker who decides to smoke only half of the amount he smoked before'. The health effects of the emissions from a steel factory's emissions are roughly one-third of the health effects of living with a smoker.

4. Discussion

We developed a method to express the health risks of long term exposure to ambient air pollution in equivalent numbers of passively smoked cigarettes in the home, based on relative risks obtained in epidemiological studies for four health outcomes reflecting childhood and adult health responses.

The equivalent number of passively smoked cigarettes ("passive cigarette-equivalents") varied within a factor three between health outcomes suggesting that an average passive cigarette-equivalent per pollutant is a useful metric. The health risk of living directly near the freeway in Amsterdam compared to a counterfactual clean situation is equivalent to daily residential ETS exposure from 10.1 cigarettes (95% CI: 6.5–13.7), more than the number of cigarettes smoked indoors at home by an average Western smoker. A local traffic measure, resulting in a 50% decline in traffic intensity in a busy inner city street in the Hague, is equivalent to the benefits of avoiding ETS from 4.3 daily smoked cigarettes in the home (95% CI: 2.5–6.1).

This method provides a tool that enables policy-makers and health professionals to express the health risks of air pollution and the health benefits of better air quality related to e.g. air quality policies in a simple, appealing manner. The method can be used both at the national/regional and at the local level, depending on the pollutant that most adequately reflects exposure. This is often PM2.5 at a larger scale and NO_2 or BC at the local scale.

The comparison between air pollution and ETS exposure was based on comparison of four health outcomes: Low Birth Weight, lung function decrements, cardiovascular mortality and lung cancer. Obviously, ETS exposure and air pollution have been linked to an increased risk of a wide range of other health outcomes (Kelly and Fussell, 2015; Öberg et al. 2010), that have not been incorporated in this study. We selected only health outcomes for which the relation with both ETS and all three pollution indicators (PM2.5, NO_2 and BC) could be quantified in meta-analyses.

The aim of our study was not to quantify the total burden of disease due to air pollution or ETS exposure (Lim et al., 2012), but to put the health risks of air pollution in perspective. It should also be noted that for both air pollution and ETS exposure, around 90% of deaths are due to cardiovascular mortality and lung cancer (WHO, 2014; Wells, 1999), two health outcomes that were incorporated in our tool.

A strength of our study is that different health outcomes were included, with different mechanistic pathways. Although different estimates were obtained per pollutant-outcome combination, which is inevitable given the inherent uncertainties of the method, most estimates had overlapping confidence intervals and varied within acceptable ranges. Summarizing the estimate for each pollutant over the health outcomes reduced the uncertainty, which is further decreased if more than one pollutant is included.

In air pollution epidemiology, one of the most intensively studied health outcomes is 'all cause mortality' (WHO, 2013; Janssen et al., 2011) which forms the basis of the calculation of Years of Life Lost (YLL) and Risk Advancement Periods (RAP). However, to our knowledge no meta-analyses are available describing the CRF between ETS exposure and all cause mortality. Therefore we incorporated cardiovascular mortality, which is responsible for 80% of total deaths due to air pollution (WHO, 2014).

4.1. Choice of passive smoking for comparison

We used passive smoking to put air pollution health risks in perspective, based upon the assumption that this risk factor is easier to understand by the public and policy makers. Passive smoking is more visible than fine particle pollution and not only associated with physiological health effects but also with annoyance. Regulating actions against smoking in public places have resulted in increased awareness of the health risk of ETS exposure and decreased acceptance for those that are involuntarily exposed to tobacco smell (Lu et al., 2011; Sivri et al., 2013). Both annoyance and (physiological) health risks contribute to lower acceptance, the relative contribution of the two is uncertain.

The prevalence of daily tobacco smoking has declined in developed countries in recent decades (Ng et al., 2014; Islami et al., 2014), but our method is – by definition – based on comparison with those that remain smoking indoors at home. However, we did not find much evidence in the literature for a decreasing trend in the exposure to ETS at home in households with passive smoking. It has also been hypothesized that smoking bans in public places resulted in more passive smoking at home. A Spanish study reported a significant reduction of 15% in self reported ETS exposure in the home among adult non-smokers comparing before (2004) and after (2012) smoke-free legislation (Sureda et al., 2014). Data from the NHANES study in the US, however, demonstrated a significant increase in serum cotinine levels for children with exposure to ETS at home between 1999 and 2012, whereas serum cotinine levels among nonsmoker adolescents with exposure to ETS at home did not change over time (Jain, 2016).

4.2. Uncertainties

This method has a number of uncertainties. One of the most important sources of uncertainty in the effect estimates is related to the choice of the risk estimates. If available, we used relative risks proposed by published systematic reviews which can be assumed to provide the most appropriate, yet imperfect, effect estimates. If those were not available, we performed additional meta-analyses based on the available published reports, providing risk estimate to the best of our knowledge.

Another important source of uncertainty is the assumption of the daily amount of cigarettes that a smoker smokes indoors at home. Our calculations were based on the assumption that the average number of smoked cigarettes was 14 per day for a smoker in Western Europe/USA (Öberg et al. 2010). No literature was available quantifying the number of cigarettes smoked indoors at home, so this was based on assumptions about the smokers time spent at home and smoking habits, as proposed by Nazaroff and Singer (2004). This resulted in the assumption of 7 daily smoked cigarettes in the home for exposure of an adult and 9 for a child. We realize this assumption is subject to error and might not

adequately reflect the average number of passively smoked cigarettes indoors at home that children and adults are exposed to. We note however that we used this assumption to calculate the effect of ETS per cigarette in previous epidemiological studies that represented residential ETS exposure as present/absent. Hence, the key issue is whether the selected numbers represented these studies well. Our method is based on comparison of health risks expressed per passively smoked cigarette and therefore, it is not sensitive to recent changes in smoking rates. However, when expressed in terms of "living with a smoker", the equivalent health effect attributed to air pollution increases with a decreasing number of cigarettes smoked indoors at home by a smoker.

To account for the uncertainty in the estimation of the daily amount of residentially smoked cigarettes we performed sensitivity analyses, evaluating different assumptions about the number of cigarettes that smokers smoke indoors at home. This illustrated that the cigarette equivalency of air pollution was linear related to the assumed number of smoked cigarettes. If the number of passively smoked cigarettes is assumed to be higher, the passive cigarette equivalents attributed to ambient air pollution is higher as well – and vice versa. The sensitivity analyses also demonstrated that, regardless the assumption of the amount smoked, the health effect of 'living near the freeway' is slightly higher than the health risk of 'living with a smoker'.

Another limitation of our study is that despite our efforts to select comparable outcomes, there were differences between the epidemiological studies of ETS and air pollution. In air pollution epidemiology, concentration response functions are available for cardiovascular mortality but fewer studies have evaluated IHD mortality. In ETS epidemiology exposure response functions are only available specifically for IHD. Since IHD is causally related to death in relation to ETS exposure and IHD is a leading cause of mortality among cardiovascular mortality, we assumed that the exposure-response functions for cardiovascular mortality hold for IHD. To test this hypothesis, we calculated the combined risk estimate for PM2.5 and IHD mortality from a review that provided risk estimates for PM2.5 and IHD mortality in a number of individual studies (Burnett et al., 2014). The combined effect for a 10 $\mu g/m^3$ increase in PM2.5 concentration and IHD mortality was 1.18 (1.05–1.33) (presented in the Supplemental Material), which is very similar to the combined effect estimate of 1.20 (1.09–1.31) for the same increase in PM2.5 concentration and cardiovascular mortality we used in our method. We did not use the IHD estimates as these are only available for PM2.5 and not for NO_2 and BC.

Furthermore, the health effect estimates for ETS in adults apply to the population that does not actively smoke, whereas in the air pollution literature the entire population is included. There is no solid evidence that allows quantitative assessment of air pollution effects in non-smoking subjects only, but there is evidence that both smokers and non-smokers are affected by air pollution (Pope et al., 2004; Dabass et al., 2016).

Another source of uncertainty is related to exposure error. Personal exposure to both air pollution and ETS is determined by activity patterns, such as time spent at home, school, work or elsewhere. The use of outdoor concentrations to assess exposure to air pollution leads to exposure misclassification. However, the concentration response functions that quantify the strength of the association between pollutant concentration and health outcomes are derived from epidemiological studies that generally are also based on outdoor concentrations. They are subject to the same degree of exposure misclassification and thus, it is justified to rely on outdoor concentrations to estimate the health effects for our comparison.

Pope et al. (2009, 2011) have evaluated exposure-response functions for PM2.5, passive and active smoking in relation to cardiovascular mortality and lung cancer. Similar to our study, IHD mortality was investigated in relation to ETS exposure and CVD mortality in relation to PM2.5 exposure. However, Pope and colleagues used ETS exposure to derive estimates of PM2.5 exposure, in order to derive the shape of the PM2.5 exposure response function. For instance, estimated average PM2.5 exposure was 30 $\mu g/m^3$ for living with a spouse who smokes, based on limited data from studies that sampled PM2.5 concentrations from ETS exposure in various setting over time (Spengler et al., 1985; Dockery and Spengler, 1981; Jenkins et al., 1996; Leaderer and Hammond, 1991). An alternative approach would be to express (changes in) PM2.5 concentration in terms of equivalent changes in concentration, caused directly by residential ETS exposure. However, the studies that sampled PM2.5 are decades old, and the strength of our method is that we compare health risks instead of various health outcomes and various pollutants.

We selected three examples with sizable contrast in air pollution exposure to illustrate the method. The impact of many local policy measures on air quality is generally more modest, since the concentrations are mainly determined at the national and European level.

Consequently, the associated gain in passive cigarette-equivalents is generally small.

We have developed a method to express health risks of air pollution in an equivalent number of passively smoked cigarettes, assuming that this is easier to communicate to the public and policy makers. Future research is needed to evaluate the usefulness of this tool for local policy makers and the public in the setting of practical environmental health problems.

5. Conclusions

We have developed a method to express the health effects of air pollution into equivalent amounts of passive smoking, based on the currently available epidemiologic evidence. The proposed method expresses the health risks of air pollution and the health benefits of better air quality in a simple, appealing manner. The method can be used both at the national/regional level and at the local level, in situations where traffic is the major source of air pollution.

Human subjects

N/A.

Animal research

N/A.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.envres.2016.04.001.

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